Cira et al v. Henry County

EXHIBIT "B"

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RE: *Cira et al v. Henry County*
DATE OF REPORT: March 7th, 2022

I. QUALIFICATIONS, RELEVANT EXPERIENCE AND CERTIFICATIONS:

I am an Emergency Medicine physician and a Medical Toxicologist certified by the American Board of Emergency Medicine in the specialties of emergency medicine and medical toxicology. I am also an Associate Professor in the Department of Emergency Medicine at the University of Texas Southwestern Medical Center at Dallas.

Additionally, I serve as Attending Physician in the Parkland Hospital Emergency Department. Parkland is a Level 1 Trauma Center and has the most emergency room visits of any single hospital in the United States. Parkland has an acute crisis psychiatric emergency room. All psychiatric patients, including those with substance abuse, must first be evaluated, stabilized, and medically cleared in the main emergency department before being transferred to the psychiatric department. As a Parkland emergency physician, I personally evaluate, manage, and treat patients presenting with trauma, medical emergencies, burns, psychiatric emergencies, overdoses and toxic exposures including illicit drugs.

I also serve as Medical Toxicology Faculty in the North Texas Poison Center. As a Medical Toxicologist, I consult with physicians in North Texas regarding the appropriate management of all types of overdoses, ingestions, and toxic exposures through the North Texas Poison Center. I am an expert in recognizing the signs and symptoms of intoxication from prescription medications, alcohol, and illicit substances.

As an Associate Professor at the University of Texas Southwestern at Dallas, I supervise medical students, interns, and residents at the bedside in the Parkland Hospital Emergency Department. I also provide lectures and training for the Department of Emergency Medicine. Through the North Texas Poison Center, I train medical students, emergency medicine residents, pharmacy residents and toxicology fellows in the management of poisoned patients as well as provide academic instruction during daily toxicology rounds.

I have cared for Dallas County's underserved population since 1999. I have helped educate and train every emergency medicine physician graduating from Parkland Hospital's emergency medicine residency program since 2002. These physicians have gone on to staff emergency departments throughout the Dallas-Fort Worth metroplex, Texas and around the United States. I have helped train every medical toxicologist graduating from Parkland Hospital's medical toxicology fellowship program since 2004. I have provided medical control to emergency medical services (EMS) from Parkland's Biotel since 1999. As an Associate Professor at the University of Texas Southwestern Medical Center in the department of emergency medicine and medical toxicology, I have remained abreast of the latest advances in evidence-based medicine

and evaluated the evidence-based medicine process that dictates the practice of the emergency medicine and medical toxicology community. For 15 years, I have provided attorneys throughout the United States and the Texas Medical Board with valuable expertise in the fields of emergency medicine and medical toxicology.

II. MATERIALS REVIEWED:

I was asked by Terry Williams from Williams, Morris, and Waymire in Buford, Georgia, to perform an independent review of the 09/20/19 incident regarding Mr. Fernando Rodriguez, hereby referred to as FR. I was provided case-specific materials and asked to render an opinion regarding impairment/intoxication as well as cause of death. My methodology involved considering evidence regarding past medical and psychiatric history, the scene investigation, case details, eyewitness testimony, pathology examination, and laboratory analysis. Those materials included:

- Officer Body-Worn Camera Videos
- Henry County Fire Department EMS Records
- Medical Examiner Report with included toxicology report
- Medical Examiner Photos
- Grady Hospital Medical Center Records
- Piedmont Healthcare Health Records
- Hampton Police Department Reports
- Henry County Police Department Records

Ultimately, I evaluated and balanced that evidence using the physician tool of "differential diagnosis" and evidence-based medicine as well as review of the medical literature.

Fernando Rodriguez (FR): DOD 09/20/2019

III. BRIEF CASE DETAILS:

- FR does not have any known prior medical history or medical records provided, age 24 at time of his death.

- 9/20/19 2100: FR and his girlfriend, Ivone Becerra, attend Imagine Music Festival near the Atlanta Motor Speedway in Hampton, GA. FR took an unknown drug that was on a square piece of paper ("or stickers") and placed in his mouth. Then he threw down his bag and phone and took off. Ms. Becerra called FR's brother and hired a Lyft driver to drive around and to find him. They were unsuccessful. Approximately 1 hour prior to police encounter, they state they lost track of him.

- 9/20/19 2205: Officer Bowlden hears and responds to dispatch call to Officer Lewis regarding a male subject (FR) walking down the middle of Oak Street in Hampton, GA naked.

- 9/20/19 2211: Officer Lewis and Officer Stroud arrive on scene and repeatedly ask FR to get on the ground, requests are ignored by FR. FR is tased and requests are made to roll over onto stomach. FR is tased 5 times while attempts are made to restrain him and requests for compliance.

- 9/20/19 2215: Officer Bowlden arrives on scene, 6th taser attempted, but not successful due to lead dislodgement. 2nd taser leads deployed by Officer Bowlden. FR continues to resist and attempts to use vehicle to stand up. Officer Lewis deploys 3rd taser leads unsuccessfully. Henry County Officer arrives and deploys 4th taser leads successfully.

- 9/20/19 2220: Hampton and Henry County officers handcuff and subdue FR with great effort from multiple officers due to FR being altered, abnormally strong, resistant to multiple bouts of taser firing, and extremely diaphoretic, per officer reports. Altercation lasted approximately 10 minutes. FR is tased 3 more times after continuing to resist and attempting to bite officers.

- 9/20/19 2226: FR seen on body camera moving head

- 9/20/19 2228: Officers first note FR is no longer breathing

- 9/20/19 2231: EMS arrives on scene, place FR on stretcher, and initiate CPR

- 9/20/19: EMS note patient in asystole, resuscitative efforts including placement of supraglottic airway result in return of spontaneous circulation for approximately 5 minutes. Rhythm recorded as PEA. Pulses lost again and patient given total of 3 rounds of epinephrine, 2mg of Narcan, return of spontaneous circulation prior to arrival in ED.

- 9/20/19 2250: FR arrives to Piedmont Healthcare. Exam shows 6 taser prongs, facial abrasions, and contusions, ecchymoses to left arm and left chest wall, abrasions to lower extremities, pupils fixed and dilated. King airway replaced with 7.5 endotracheal tube. Vitals upon arrival showed BP 106/46, Pulse 135, Temperature 103 F.

- 9/20/19: Labs show leukocytosis, normal hemoglobin, normal cardiac enzymes, elevated coagulation markers, decreased kidney function. EKG does not show STEMI, chest x-ray without significant abnormality. Noted to be spontaneously opening eyes prior to transfer.

- 9/21/19: Transferred to Grady Hospital in Atlanta, GA for higher level of care. CT/Trauma work up unremarkable, found to have metabolic derangements including significant acidosis and hyperkalemia.

- 9/23/19: FR remained in critical condition progressing to disseminated intravascular coagulation requiring blood pressure support, dialysis, respiratory support, and aggressive resuscitation efforts. Condition continued to deteriorate until PEA arrest without

successful resuscitation following multiple rounds of ACLS. Time of death at 0143 on 9/23/19. Pronounced by Kirsten Mary Beacher, MD.

- 9/24/19: Autopsy of Fernando Rodriguez performed at 0830 by Steven P. Atkinson, MD at the Georgia Bureau of Investigation in Decatur Georgia with the following findings:

SUMMARY OF FINDINGS:

I. Asphyxia:
   A. Circumstances of cardiopulmonary arrest during restraint in the prone position with compression of the chest.
   B. Resuscitation with 3-day hospitalization before being pronounced dead.
   C. Bilateral patchy conjunctival hemorrhage.

II. Blunt trauma of the head, torso, and extremities:
   A. Abrasions and contusions of face.
   B. Abrasions of torso.
   C. Contusions of upper extremities.
   D. Abrasions of lower extremities.
   D. Soft tissue hemorrhage of back, upper extremities, and lower extremities.

III. Toxicology, admission blood:
   A. Positive, lysergic acid diethylamide (LSD), 1.0 ng/mL (testing performed at NMS Labs, Horsham, PA).
   B. Negative for ethanol.
   C. Negative comprehensive drug screen.

IV. Status post taser use during altercation:
   A. Healing taser probe injuries identified on abdomen, left thigh, and right thigh.

V. Hypertensive and atherosclerotic cardiovascular disease:
   A. Cardiomegaly; heart weight is 560 grams.
   B. Left ventricular hypertrophy.
   C. Coronary artery disease.

VI. Bilateral pleural fusions and anasarca consistent with hospitalization.

VII. Vitreous electrolytes not contributory to death.

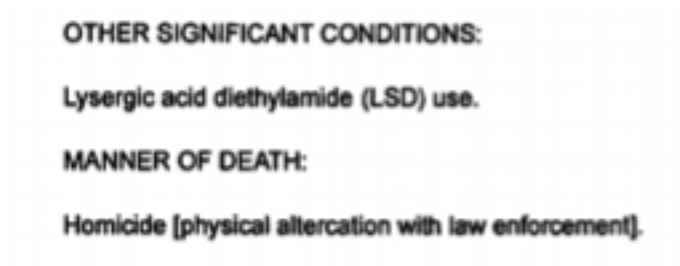
COMMENT / OPINION:

This 24-year-old Hispanic male died as a result of asphyxia due to physical restraint in the prone position with compression of the chest. Another significant condition contributing to his death was lysergic acid diethylamide (LSD) use. He was involved in a physical altercation with law enforcement personnel. During this altercation, handcuffs were placed around his wrists and leg shackles were placed around his ankles. He was then placed in the prone position with his arms above his head. One officer stood on the handcuffs and a second officer stood on the leg shackles restraining him. A third officer then placed his knee on the decedent's back applying pressure to his chest. During this time, the decedent stopped breathing and became pulseless. The decedent was subsequently resuscitated and hospitalized for 3 days before being pronounced dead. Based on the information known at this time, the manner of death is classified as homicide.

CAUSE OF DEATH:

Asphyxia due to physical restraint in prone position with compression of chest.

- Toxicology testing revealed:



OTHER SIGNIFICANT CONDITIONS:

Lysergic acid diethylamide (LSD) use.

MANNER OF DEATH:

Homicide [physical altercation with law enforcement].

IV. DISCUSSION:

When evaluating cause of death, a systematic approach is necessary to first rule-out obvious causes of death from trauma or natural causes. Sudden cardiac arrest from an underlying arrhythmia was also considered. An analysis of FR's death from a toxicological perspective was performed. FR's death was then evaluated based on literature regarding sudden in-custody deaths including "positional asphyxia", death from conducted energy weapons (CEWs), excited delirium syndrome (ExDS) and stress cardiomyopathy.

A. TRAUMATIC CAUSE OF DEATH

During FR's encounter with the police on 09/20/2019 there is no mention of signs of prior trauma to FR's body. FR was resisting, fighting, thrashing on concrete without any clothing. It required significant effort from multiple officers to subdue FR, causing him to sustain multiple abrasions and lacerations to his face, chest, back, and extremities, which were documented in the hospital as well. These external signs of minor trauma at autopsy were noted, but superficial lacerations, abrasions or contusions do not cause death in and of themselves. Autopsy revealed minor to moderative evidence of external traumatic injuries including facial abrasions/contusion, abrasions to the extremities, abrasions and hemorrhage along the back. Bilateral patchy conjunctival hemorrhage is most consistent with post mortem changes. Autopsy did not reveal any underlying organ injuries from blunt trauma in the thorax or intra-abdominal injuries such as liver or spleen lacerations. The pleural, pericardial, and peritoneal spaces did not reveal any major hemorrhage. Autopsy included dissection of the neck, with layer-by-layer examination was grossly unremarkable for trauma. The hyoid bone, larynx, and cervical spine were indicated by the Medical Examiner as grossly intact. The Medical Examiner also noted that the soft tissues of the neck, including the skeletal muscles, blood vessels, thyroid, and connective tissues were grossly unremarkable. There were no skull fractures seen at autopsy. Trauma to the head or face can cause an epidural hematoma, subdural hematoma, or subarachnoid hemorrhage—all of which could cause sudden death. However, none of these findings were present at autopsy. **Therefore, FR did not die from traumatic causes resulting from any supposed excessive use of force by any law enforcement officer involved in the incident of 09/20/2019.**

B. NATURAL CAUSES OF DEATH WITH FINDINGS ON AUTOPSY

• Table 1 lists frequent causes of adult sudden death from natural causes.

Table 1. Frequent Natural Causes of Adult Sudden Death

Airway	Choking/acute airway obstruction
Pulmonary	Acute asthma exacerbation
	Pulmonary embolism
Neurological diseases	Sudden unexpected death in epilepsy
	Acute CNS hemorrhage
Cardiovascular diseases	Coronary artery disease (CAD)
	Hypertrophic cardiomyopathy
	Dilated cardiomyopathy
	Lymphocytic myocarditis
	Aortic stenosis
	Congenital cardiac abnormality
	Coronary artery dissection
	Aortic dissection

The autopsy did not reveal any signs of airway obstruction from a foreign body or otherwise. FR did not have a known medical history of asthma. Pulmonary embolism is not an uncommon cause of sudden death. Pulmonary embolism is a blood clot originating from the deep veins (usually of a lower extremity) and will "travel" to the vasculature of the lungs. A pulmonary embolism was not found on autopsy. There were no descriptions of FR developing seizure activity before he became unresponsive, and this is not evident on the police body camera video either. There were no intra-cerebral hemorrhages found on postmortem examination of the brain. FR was found to have mild to moderate atherosclerosis in the coronary arteries, with no areas of infarct. There was also concentric left ventricular hypertrophy. These findings do not alone explain death. **Thus, there were no underlying gross abnormalities revealed by FR's autopsy or post-mortem histological examination that explain his sudden death.**

C. ARRHYTHMIA

An arrhythmia is an electrical disturbance of the heart. A lethal arrhythmia can cause sudden cardiac arrest and death. Abnormal heart rhythms that originate in the lower part of the heart are called ventricular arrhythmias. Ventricular arrhythmias include ventricular tachycardia and ventricular fibrillation. All other cardiac arrest rhythms (meaning rhythms that do not have a pulse) that fall outside the category of pulseless ventricular tachycardia, ventricular fibrillation, or asystole (flat line) are considered pulseless electrical activity (PEA). Warning signs for sudden death include history of sudden unexplained death of a family member under age 40, fainting or seizure during exertion or excitement, or consistent chest pain or shortness of breath during exercise. Unfortunately, an arrhythmia cannot be detected after death since the heart must be electrically active to diagnose an arrhythmia. An electrocardiogram (EKG) can confirm an arrhythmia in living patients, but not in dead patients since there is no longer any electrical activity. There are gross cardiac abnormalities—including hypertrophic cardiomyopathy, dilated cardiomyopathy, congenital abnormalities, etc. as listed in the table above-- that can be

associated with an arrhythmia. These findings to not "prove" that death occurred from a lethal arrhythmia as there are many individuals who are alive with these conditions. And individuals with structurally normal hearts still can die from sudden cardiac arrest. Structural cardiac abnormalities noted on autopsy and discovered incidentally do not prove that a lethal arrhythmia occurred and caused sudden death. **Therefore, attributing the cause of death to an arrhythmia is a diagnosis of exclusion**.

D. TOXICOLOGICAL CAUSE OF DEATH

When the gross post-mortem examination does not reveal a cause of death, it is necessary to next review the toxicology testing. When determining a potential toxicological cause of death, the substances found in the blood or the vitreous humor are most likely to have exerted effects at or around the time of death. However, impairment, intoxication, and cause of death are not determined by drug concentrations alone. Behavioral observations and/or perimortem circumstances must be correlated with drug concentrations to determine impairment, intoxication, or cause of death. Furthermore, persons abusing dangerous drugs do not need to "overdose" on these substances to die. "Therapeutic" amounts of illicit drugs can undoubtedly cause death. Additionally, drug dealers do not have good quality control so there is no method for determining the amount of an illicit drug that was ingested. A drug concentration, however, confirms the presence of that substance with no chance of a false positive result. Behavioral observations and/or perimortem circumstances must be correlated with drug concentrations to determine impairment, intoxication, or cause of death.

i. General Interpretation of Drug Levels.

Clinical impairment/intoxication and/or cause of death are not defined by drug levels alone—whether in the antemortem or postmortem setting. In other words, drug levels must not be interpreted "in a vacuum." Tolerance, sex, drug interaction, and mode of use can confound interpretation of drug levels. Behavioral observations and/or circumstances surrounding an incident or death must be correlated with confirmation of the substance.

ii. Lysergic acid diethylamide (LSD) Overview

Lysergic acid diethylamide (LSD) is classified as a hallucinogen, psychedelic, or psychostimulant that was first synthesized in 1938. It is manufactured from lysergic acid, a naturally occurring substance found in ergot fungus that grows on plants. It is a Schedule I controlled substance which affects multiple neurotransmitter receptors in the cortex of the brain, including serotonin, dopamine, and glutamate. LSD is usually taken orally in the form of liquid, powder, tablets, or ***applied to paper squares (known as blotter papers)***, among other formulations. It was marketed as an adjunct for analytic psychotherapy and was also used by the CIA in the 1960s as an interrogation and mind control aid. LSD was popularized as a recreational drug of abuse by Dr. Timothy Leary with his exhortation to "tune in, turn on, and drop out." By the 1980s, use of LSD had markedly decreased because of popular and literature reports suggesting that LSD could cause brain damage, including "bad trips" and flashbacks that could occur years after use.[1]

iii. Clinical Effects of LSD

Due to its interaction with neurotransmitter receptors, clinical effects begin to occur within 30-60 minutes after ingestion, with a typical duration of 10-12 hours. Objective signs of LSD use may include a fast heartbeat (tachycardia), enlarged pupils (mydriasis), high blood pressure (hypertension), fast breathing (tachypnea), an elevated temperature (hyperthermia), seizures, and increased sweating (diaphoresis). These physical signs typically precede the altered perceptual symptoms experienced by users.[1]

Subjective symptoms of LSD ingestion include a sense of heightened awareness of visual and auditory stimuli accompanied by auditory and visual hallucinations, along with confusion of the senses (synesthesia) and distortions of size, shape, and color. Users may also have a sense of enhanced insight or awareness, or a feeling of depersonalization. [1,2] In some cases, users may experience a "bad trip," which can lead to anxiety, combativeness, and bizarre behavior, including violence toward oneself or others. [1,2,3]

Long-term use of LSD can cause prolonged psychotic reactions, severe depression, and exacerbation of the user's preexisting psychiatric illness. Users may have panic attacks, disabling flashbacks (hallucinogen persisting perception disorder), or extended psychosis that requires hospitalization.[1,2,3] The reported percentage of users who experience flashbacks has varied widely from 15-80%.[1] Risk factors for precipitating a flashback can include anesthesia, alcohol use, medication use, stress, illness, sleep deprivation, and exercise. [1,4] There also appears to be a relationship between the incidence of flashbacks and the frequency or length of time of LSD use.[2] It is unclear if FR had used LSD previously or frequently based on the information I was provided.

LSD alone is not known to have directly toxic deadly affects, but rather users may induce a psychotic state where they can pose a danger to themselves or others.[5] There are many cases reports of LSD induced psychosis causing a person to be a danger to themselves or others in a state of altered perception. In the case of FR, he was walking on the street, naked, without regard for the danger around him including vehicles, but also was combative and aggressive with officers despite multiple attempts at verbal de-escalation.

iv. LSD Levels in Fernando Rodriguez

FR took a drug on a square piece of paper or "stickers" called blotter papers. LSD is typically sold on these blotter papers. FR's behavior and appearance on 09/20/2019 is consistent with his use of LSD and he was obviously intoxicated by LSD. FR's LSD concentration was 1 ng/mL. **FR's LSD concentration certainly falls within the reported range of LSD concentrations shown to have clinical effects including altered sensorium, fever, tachycardia, hallucinations. FR was indeed intoxicated by LSD. LSD intoxication, stress and prolonged exertion may have contributed to his death. The exact nuances of FR's perception, sensorium and depersonalization will never be known. Regardless of his ability or inability to process the events of 09/20/2019, FR was clearly a danger to others. The correlation of LSD use and ExDS causing death is evident in the medical literature.**

E. SUDDEN IN-CUSTODY DEATH

"Sudden in–custody death syndrome" has been used to describe unexplained deaths when police are involved and is important to consider in FR's sudden cardiac arrest. Encompassed in this syndrome are also the terms "excited delirium syndrome" (ExDs), "capture cardiomyopathy" and "positional asphyxia"

i. Excited Delirium:

Physicians that do not practice in an emergency department setting will likely never encounter ExDS. For example, ExDS patients do not present to community internists, surgeon's offices, or general medical floors. But ExDS is a syndrome observed by and managed by law enforcement, first responders, and emergency physicians. The American College of Emergency Physicians (ACEP) formally recognized ExDS in 2009. ACEP convened a task force that performed an extensive review of the literature and ultimately published a position paper on ExDS. Position papers hold great weight in the medical community because they are not written by a single author or small group of authors but represent a consensus of a task force and require agreement by all the authors on the final paper. In the position paper, the ACEP task force concluded: "Based upon available evidence, it is the consensus of the American College of Emergency Physicians Task Force that Excited Delirium Syndrome is a real syndrome with uncertain, likely multiple, etiologies."[6] In April 2011, the National Institute of Justice ("NIJ") of the United States (US) Department of Justice ("DOJ") convened a meeting of experts in ExDS known as the ExDS Workshop Panel. A report was then published that discussed the definition, epidemiology, pathophysiology, differential diagnosis, and acute treatment of ExDS. The features of ExDS are listed below.[7] The features of ExDS that were also exhibited by FR are checked:

ExDS Features in History

√ Male gender
Mean age in the 30s
√ Sudden onset
√ History of psychostimulant abuse
History of mental illness

ExDS Features Evident at the Scene

√ Call for disturbance/psychomotor agitation/excitation
√ Violent/combative/belligerent/assault call
√ Not responding to authorities/verbal commands
√ Psychosis/delusional/paranoid/fearful
√ Yelling/shouting/guttural sounds
√ Disrobing/inappropriate clothing
√ Violence toward/destruction of inanimate objects
√ Walking/running in traffic
√ High body mass index

Features Evident on Police Contact

√ Significant resistance to physical restraint
√ Superhuman strength
√ Impervious to pain
√ Continued to struggle despite restraint
√ Profuse sweating/clammy skin

ExDS Features with Clinical Assessment

Tachypnea
√ Tachycardia
√ Hyperthermia
Hypertension
√ Acidosis
Rhabdomyolysis

ExDS Features of Death

√ Period of tranquility "giving up"
√Sudden collapse after restraint
√ Respiratory arrest described
√ Cardiac rhythm brady-asystole/PEA
√ Aggressive resuscitation unsuccessful

ExDS Features on Autopsy

√ Drug screen positive for psychostimulants
√ Drug levels lower than anticipated
√ No anatomic correlate for death
Dopamine transporter dysregulation [not assessed]
√ Cardiac hypertrophy

ExDS is precipitated by mental illness, recent psychostimulant use or both. Psychostimulants often include cocaine, amphetamines, or phencyclidine("PCP"). ExDS is typically a retrospective diagnosis and is often difficult for physicians to make contemporaneously merely from observation of the agitated individual. Thus, it is very difficult if not impossible for law enforcement or first responders to differentiate ExDS from alcohol or drug intoxication, mental illness, or mere combativeness. There is concern that FR was abusing LSD. In her October 2016 article, Deborah Mash explains how all psychostimulants, such as LSD, increase the synaptic levels of dopamine, which may explain why chronic psychostimulant abusers are at risk for exhibiting the behavioral symptoms associated with ExDS. Finally, Mash concludes[8]:

> *Elevated levels of dopamine coupled with failed dopamine transporter function leads to agitation, paranoia, and violent behavior associated with ExDs. Increased dopamine levels also affect heart rate, respiration, and temperature control with elevation resulting in tachycardia, tachypnea, and hyperthermia. Hyperthermia is a hallmark of excited delirium and a harbinger of death in this syndromal disorder. Victims of excited delirium*

> *are in an extremely heightened emotional state with marked paranoia and mounting irrational fear. Central neuronal circuitry in the brain-heart axis may be a precipitant of sudden fatal arrhythmia, since hyperdopaminergic signaling in the limbic system translates extreme emotional stress into autonomic toxicity and the demise of the heart. The connection between hyperdopaminergia in ExDs and chaotic signaling in these higher brain autonomic regulatory centers may explain the abrupt loss of autonomic function that leads to sudden unexpected death of these victims. Excited delirium is a syndromal disorder, which is controversial and highly debated because the mechanism is lethality is unknown. Molecular studies of the brains of autopsy victims who died in states of excited delirium reveal the loss of dopamine transporter function as a possible trigger of a cascade of coordinated neural activity that contributes to asphyxia and sudden cardiac arrest.*

ExDS is a syndromic disorder. As with many other clinical syndromes, there is not a definitive diagnostic "test" for ExDS and must be identified by its clinical features. Not all features need to be present to reach a diagnosis of ExDS. As with other clinical syndromes, ExDS exists along a spectrum with minor ExDS displaying a few features and severe ExDS displaying many of the features.

FR's behavior the evening 09/20/2019 is consistent with LSD intoxication. His interaction with police officers displayed erratic, aggressive behavior, with increased strength, resistance to taser effects, and diaphoresis. He was also noted to be significantly febrile upon arrival to the ED. FR had already "moved along the spectrum" of agitation to reach delirium.

When FR suffered sudden collapse and cardiac arrest, the initial rhythm documented by EMS was pulseless electrical activity (PEA)—not a ventricular arrhythmia. This is an important feature that distinguishes death from a cardiac arrhythmia due to LSD intoxication. Individuals that are LSD toxic may not necessarily display ExDS. Individuals that display ExDS from LSD are also LSD toxic. **FR's death is consistent with LSD-induced ExDS.**

ii. Positional Asphyxia:

Asphyxia is a restriction of breathing that can interfere with the delivery of oxygen to tissue. Discussion regarding asphyxia is often hindered by confusion as there is not standardization of the definitions or classification of asphyxia deaths. Sauvageau et al. proposed classifying asphyxia into four main categories: suffocation, strangulation, mechanical asphyxia, and drowning. Mechanical asphyxia includes positional asphyxia and traumatic asphyxia. Positional asphyxia is hypothesized to cause death when an individual is placed in the prone (or face-down) position. Historically, positional asphyxia was used to describe deaths in individuals who had happened into odd positions where their chest had been splinted or the diaphragm was prevented from moving. The respective definitions are given in the table below[9]:

TABLE 6—*Definitions of terms in the proposed unified classification.*

Term	Definition
Suffocation	A broad term encompassing different types of asphyxia such as vitiated atmosphere and smothering, associated with deprivation of oxygen
Smothering	Asphyxia by obstruction of the air passages above the epiglottis, including the nose, mouth and pharynx
Choking	Asphyxia by obstruction of the air passages below the epiglottis
Confined spaces/ entrapment/ vitiated atmosphere	Asphyxia in an inadequate atmosphere by reduction of oxygen, displacement of oxygen by other gases or by gases causing chemical interference with the oxygen uptake and utilization
Strangulation	Asphyxia by closure of the blood vessels and/or air passages of the neck as a result of external pressure on the neck
Ligature strangulation	A form of strangulation in which the pressure on the neck is applied by a constricting band tightened by a force other than the body weight
Hanging	A form of strangulation in which the pressure on the neck is applied by a constricting band tightened by the gravitational weight of the body or part of the body
Manual strangulation	A form of strangulation caused by an external pressure on the structures of the neck by hands, forearms or other limbs
Mechanical asphyxia	Asphyxia by restriction of respiratory movements, either by the position of the body or by external chest compression
Positional or postural asphyxia	A type of asphyxia where the position of an individual compromises the ability to breathe
Traumatic asphyxia	A type of asphyxia caused by external chest compression by a heavy object
Drowning	Asphyxia by immersion in a liquid

Thus, mechanical asphyxia involves restriction of respiratory movements, either by position of the body or by external chest compression. Positional or postural asphyxia connotes that the individual's position compromises the ability to breathe. Traumatic asphyxia is caused by external chest compression by a heavy object. FR was continuing to resist, move, and grunt during his struggle with police indicating that he was breathing. Not infrequently, facial plethora (redness of the face) will be noted on autopsy when an individual has died from asphyxiation. There were no autopsy findings to suggest asphyxia on FR's autopsy report and the images of the conjunctival findings of the eyes seem more consistent with postmortem change as opposed to asphyxia.

More importantly, current literature does not support the mechanism of officer-induced positional asphyxia, especially simply from prone positioning. Let's review a sampling of the most recent literature. In 2007, Michaelwitz et al. investigated the ventilatory and metabolic demands in healthy adults who had been placed in the prone maximal restraint position. The maximal minute ventilation was measured. They concluded that the decrease in maximal minute ventilation was not clinically significant in these subjects and that they were still able to supply their ventilatory needs[7] In 2012, Hall et al. concluded that the prone position had no clinically significant effects on subject physiology.[10] In 2013, Savaser et al. evaluated the effect of maximal prone restraint on subjects aged 22 to 42 years old. Volunteers were hogtied in the supine, prone, prone maximal restraint with no weight force, and prone maximal restraint with 50 pounds added to the subject's back, and prone maximal restraint with 100 pounds added to the subject's back for 3 minutes. There were no statistical differences in heart rate, mean arterial pressure (MAP), and oxygen saturation in the variety of positions.[11] In 2014, Sloane found no evidence of hypoxia or hypoventilation in 10 intensely exercising volunteers who were then placed in prone maximal restraint in 3 different positions for 15 minutes.[12] Finally in 2016, Karch nicely explores alternative theories in arrest-related deaths.[13] **Therefore, it is not within reasonable medical probability that FR died from mechanical, positional, postural, traumatic or restraint asphyxia.**

iii. Stress Cardiomyopathy:

Stress cardiomyopathy, or "capture myopathy", is a fatal stress reaction characterized by acidosis and rhabdomyolysis followed by death within minutes to weeks. This condition was first defined

in the 1950's in animal models that is observed across different species. The pathophysiology behind stress cardiomyopathy is a combination of fear, sympathetic nervous system activation the endocrine/adrenal system coupled with muscular activity. Acute stress as the fatal mechanism that occurs in a short time span from exposure to death include "Capture Shock Syndrome" (CSS) and "Delayed Per Acute Syndrome" (DPS). In animal models, animals displaying CSS die either while restrained or up to 6 hours later. During this time frame, the animals are seen trying to escape followed by lying motionless. During this motionless period the animals are found to have shallow rapid breathing, hyperthermia, tachycardia and a weak pulse. DPS is characterized by animals having an initial stress exposure, a brief episode of trying to escape, followed by becoming motionless and in cardiac arrest. These cardiac arrests are typically bradyasystolic arrests. Animals that experienced CSS or DPS were found to have elevated liver enzymes and creatinine kinase circulating in their blood. A summary of clinical and laboratory findings can be seen below[14]:

	Capture myopathy	
	CSS	DPS
Timespan	Hours	Minutes
Heart rate just before death	Tachycardia	Ventricular fibrillation
Blood pressure	Hypotension	–
Breathing	Fast Shallow	–
Pulse	Weak	–
Temperature	Hyperthermia	
Sweat	Diaphoresis	
Catecholamine	High serum level	
Creatinine kinases	High Some with Rhabdomyolysis	
Dopamine	–	
Damage on cardiac muscle tissue	Multiple in CSS, not DPS CBN near cardiac autonomic nerve endings in animal stress testing	
Behavior	Extreme activity - Screaming - Seemingly immune to pain Later still	

An explosive autonomic system (faster animals who can accelerate quickly, larger brains) was found to be a risk factor for stress cardiomyopathy. Translating this information to humans, repeated sympathomimetic use certainly upregulates the autonomic nervous system.

Sudden cardiac death (SCD) is well documented, particularly in males undergoing psychological stress. In a study in 2015, autopsies of young (<36 years of age) males who experienced SCD showed that 60% of these individuals had morphologically normal hearts.[15] 31% of deaths in this study were in the setting of physical restraint. **Stress cardiomyopathy cannot be ruled out as a cause or contributor to FR's death.**

F. CEW (TASERS)

Tasers primarily have two modes: "drive stun" mode and "probe" mode. Drive-stun mode uses less quantum of force than probe mode and is used for pain compliance. Drive-stun mode does not cause neuromuscular incapacitation. However, even if the taser was used in probe mode, it would not cause cardiac arrest. A review of basic electrical principles is essential to understand electrical injury and how electricity might cause a fatal arrhythmia. These principles are also critical to understand how CEWs affect the human body.

At Parkland, we receive all burn/electrical injuries around North Texas. I evaluate and treat many patients with electrical injuries. Type and amount of delivered electrical current (or charge), current pathway, resistance to flow and duration of exposure all determine the severity of an electrical injury. Reports in the media often focus on the fact that the CEW's output is 50,000 volts. Voltage alone is not an indicator of electrical safety or risk of electrical injury. Whereas 50,000 volts may sound impressive, static electricity can exceed 30,000 volts. A Van der Graaf generator ranges from 100,000 to 25 million volts—yet children safely, without risk of injury, place their hands on them in science museums and science classrooms all the time. As previously stated, the severity of electrical injury does not depend upon the voltage but other factors. Static electricity, Van der Graaf generators, and tasers all have low current—and that is why, in part, they are not dangerous. Cardiac capture is when an electrical stimulus causes a corresponding electrical depolarization of the atria and ventricles of the heart. This then must be followed by mechanical capture where the heart actually "beats" in response to this depolarization. There is a complex relationship between current, voltage, resistance and sufficiently rapid, or prolonged, cardiac capture causing a fatal rhythm. Another important relationship to recognize is the relationship between electricity and defibrillation in terms of joules (J). That is because defibrillators in the medical setting to convert patients out of VF require 200 J to 360 J. For pediatric defibrillation the standard is 2–9 J/kg (joules/kilogram) for infants and children. Thus, for a 5 kg infant Pediatric Advanced Life Support ("PALS") Guidelines states that 10–45 J have been found effective "with negligible adverse effects." Also, the surface area of defibrillator paddles is significantly larger than the amount of area covered by CEW probes.

See the illustrations below:

Figure 1 Defibrillator paddles.

Figure 2 13-millimeter (mm) TASER probes

Merely understanding basic electrical principles, recognizing the small joule delivered energy by the CEW, and the small surface area of a TASER probe, one can visualize how highly unlikely a CEW delivered charge would obtain cardiac capture sufficiently, rapidly, or for enough amount of time to induce a fatal rhythm. Cardiac capture in and of itself is not injurious. Cardiac capture is not synonymous with VF or cardiac arrest. As all emergency physicians know, and as clearly crystallized in literature, there is a significant safety margin between cardiac capture and sufficiently rapid or prolonged cardiac capture to induce VF or cardiac arrest. Emergency physicians often utilize transthoracic cardiac pacemakers that deliver many times more charge than a CEW without concern for inducing VF or cardiac arrest because of the significant safety margin. Besides understanding basic electrical principles and heart electrophysiology when assessing allegations of cause of death from a CEW, it is also important to carefully review at least a sampling of the current literature. Jauchem concluded in the Journal of Forensic and Legal Medicine[16]:

> *"Primary effects of factors coincident with ECD [electronic control device] exposure events may, by themselves, be more harmful than effects of limited ECD applications. Many victims of drug-induced excited delirium die without the application of any specific law-enforcement techniques. Stone noted that, on the basis of medical evidence, ECDs are not "the causes, in and of themselves", of sudden deaths in custody".*[17]

Ideker and Dosdall published a review article in 2007 in The American Journal of Forensic Medicine and Pathology. They reviewed the available scientific literature and concluded that the immediate induction of VF by the direct electrical effects of the TASER X26 CEW on the normal adult heart is unlikely and that the induction of delayed cardiac arrest by this mechanism is extremely unlikely.[18] Other articles in the medical literature studied the effects of the TASER X26 CEW on the heart. Ultimately, it was concluded that the TASER X26 CEW produced no significant electrocardiograph (EKG) changes and no detectable dysrhythmias.[19,20,21] If one is struck by lightning and suffers a ventricular fibrillation arrest, **the arrhythmia occurs immediately on contact with the electric "jolt"**— not seconds to minutes later. Nevertheless, once one understands basic electrical principles and reviews the literature and applies that research and literature to the facts of the FR incident, **it is implausible that FR died from the CEW applications by police officers.**

V. OPINIONS:

1. LSD intoxication along with prolonged exertion contributed to FR's death. Also, FR displayed many features consistent with ExDS. Therefore, FR died from LSD-induced ExDS on 09/20/2019. Stress cardiomyopathy cannot be ruled out as a contributor.

2. FR did not die from traumatic causes due to supposed excessive use of force by the police officers on 09/20/2019.

3. FR did not die from CEW use.

4. FR did not die from mechanical, positional, postural, traumatic, or restraint asphyxia.

The opinions reached in this report are based on my education, training, and experience. These conclusions are all within reasonable medical certainty and/or probability. I reserve the right to amend this report should further information become available.

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